☐ 1: NM_002888. Homo sapiens reti...[gi:4506424] Links

LOCUS RARRES1 846 bp mRNA linear PRI 23-DEC-2002
DEFINITION Homo sapiens retinoic acid receptor responder (tazarotene induced) 1 (RARRES1), mRNA.
ACCESSION NM_002888
VERSION NM_002888.1 GI:4506424
KEYWORDS .
SOURCE Homo sapiens (human)
ORGANISM Homo sapiens
Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Primates; Catarrhini; Hominidae; Homo.
REFERENCE 1 (bases 1 to 846)
AUTHORS Nagpal,S., Patel,S., Asano,A.T., Johnson,A.T., Duvic,M. and Chandraratna,R.A.
TITLE Tazarotene-induced gene 1 (TIG1), a novel retinoic acid receptor-responsive gene in skin
JOURNAL J. Invest. Dermatol. 106 (2), 269-274 (1996)
MEDLINE 96179739
PUBMED 8601727
REFERENCE 2 (bases 1 to 846)
AUTHORS Duvic,M., Nagpal,S., Asano,A.T. and Chandraratna,R.A.
TITLE Molecular mechanisms of tazarotene action in psoriasis
JOURNAL J. Am. Acad. Dermatol. 37 (2 Pt 3), S18-S24 (1997)
MEDLINE 97416602
PUBMED 9270552
COMMENT REVIEWED REFSEQ: This record has been curated by NCBI staff. The reference sequence was derived from U27185.1.
Summary: Retinoids exert biologic effects such as potent growth inhibitory and cell differentiation activities and are used in the treatment of hyperproliferative dermatological diseases. These effects are mediated by specific nuclear receptor proteins that are members of the steroid and thyroid hormone receptor superfamily of transcriptional regulators. RARRES1, RARRES2, and RARRES3 are genes whose expression is upregulated by the synthetic retinoid tazarotene. RARRES1 is thought to act as a putative adhesion molecule or cell surface receptor.
COMPLETENESS: complete on the 3' end.
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Revised: July 5, 2002.

Feb 4 2003 11:22:44

1: J Invest Dermatol 1996 Feb;106(2):269-74

Tazarotene-induced gene 1 (TIG1), a novel retinoic acid receptor-responsive gene in skin.

Nagpal S, Patel S, Asano AT, Johnson AT, Duvic M, Chandraratna RA.

Retinoid Research, Departments of Biology and Chemistry, Allergan Incorporated, Irvine, CA 92713-9534, USA.

Retinoids exert their effect through ligand-dependent transcription factors, retinoic acid receptors (RARalpha, beta, and gamma) and retinoid X receptor (RXRalpha, beta, and gamma), which belong to the superfamily of steroid/thyroid/vitamin D3, nuclear receptors. Using a subtraction hybridization approach, we have identified a cDNA sequence, Tazarotene Induced Gene 1 (TIG1), which is highly upregulated in skin raft cultures by an RARbeta/gamma - selective retinoid AGN 190168 (tazarotene/ethyl 6-[2-(4,4-dimethylthiochroman-6-yl)-ethynyl]nicotinate), which is effective in the treatment of psoriasis. The retinoid-mediated upregulation in the expression of TIG1 was confirmed by Southern and Northern analyses. Upon sequencing, TIG1 was found to be a novel cDNA which encodes a protein of 228 amino acids whose sequence suggests that is a transmembrane protein with a small N-terminal intracellular region, a single membrane-spanning hydrophobic region, and a large C-terminal extracellular region containing a glycosylation signal. We demonstrate that TIG1 is also upregulated by AGN 190168 in skin raft cultures prepared from psoriatic fibroblasts and normal keratinocytes and in primary fibroblast and keratinocyte cultures. We also show that TIG1 is upregulated by retinoic acid receptor but not by retinoid X receptor-specific synthetic retinoids. Finally, we demonstrate that TIG1 is induced by AGN 190168 in psoriatic lesions during the course of clinical treatment of the disease.

MeSH Terms:

- Amino Acid Sequence
- Base Sequence
- Biopsy
- Gene Expression/drug effects
- Human
- In Situ Hybridization/methods
- Molecular Sequence Data
- Nicotinic Acids/pharmacology*
- Psoriasis/physiopathology
- Psoriasis/pathology
- Psoriasis/genetics
- Receptors, Retinoic Acid/genetics*
- Skin/chemistry
- Skin Physiology*
- Tissue Culture
- Up-Regulation

Substances:

- tazarotene
- Receptors, Retinoic Acid
- Nicotinic Acids

Secondary source id:

- GENBANK/U27185

PMID: 8601727 [PubMed - indexed for MEDLINE]

09/865,879

Your SELECT statement is:
s urokinase and retinoid

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4	71: ELSEVIER BIOBASE_1994-2003/Feb W2
23	73: EMBASE_1974-2003/Feb W2
4	94: JICST-EPlus_1985-2003/Nov W3
6	144: Pascal_1973-2003/Feb W1
2	149: TGG Health&Wellness DB(SM)_1976-2003/Jan W4
10	155: MEDLINE(R)_1966-2003/Feb W2
2	156: ToxFile_1964-2002/Nov W3
6	159: Cancerlit_1975-2002/Oct
7	399: CA SEARCH(R)_1967-2003/UD=13807
2	442: AMA Journals_1982-2003/May B1
1	444: New England Journal of Med._1985-2003/Feb W3

File 5:Biosis Previews(R) 1969-2003/Feb W2
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***File 34: Alert feature enhanced for multiple files, duplicates** removal, customized scheduling. See HELP ALERT.
File 73:EMBASE 1974-2003/Feb W2
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File 155:MEDLINE(R) 1966-2003/Feb W2
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S2	42488	RETINOID?
S3	109	S1 AND S2
S4	81	S3 NOT PY=>2000
S5	42	RD (unique items)
S6	28	S5 AND EXPRESS?

6/9/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12320514 BIOSIS NO.: 200000041542
Retinoic acid stimulates plasminogen activator inhibitor 2 production by blood mononuclear cells and inhibits urokinase **-induced extracellular proteolysis.**
AUTHOR: Montemurro P; Barbuti G; Conese M; Gabriele S; Petio M; Colucci M; Semeraro N(a)
AUTHOR ADDRESS: (a)Dipartimento di Scienze Biomediche e Oncologia Umana, Sezione di Patologia Generale, Universita-Policlinico, Piazza G. Cesare, I-70124, Bari**Italy
JOURNAL: British Journal of Haematology 107 (2):p294-299 Nov., 1999
ISSN: 0007-1048
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: **Retinoids** have been shown to modulate several functions of mononuclear phagocytes. We investigated the in vitro effect of all-trans-retinoic acid (ATRA) on the production of two major fibrinolytic components, **urokinase** -type plasminogen activator (u-PA) and PA inhibitor 2 (PAI-2), by human blood mononuclear cells (MNC). ATRA caused a dose-dependent (range 0.01-10 muM) accumulation of PAI-2 antigen and activity into the cell culture medium, with a maximal increase (about 5-fold over control) at a concentration of 1-10 muM. Similarly, a dose-dependent increase in PAI-2 antigen was observed in cell extracts upon ATRA stimulation. Northern blot analysis showed a parallel increase

in the amount of PAI-2 mRNA in ATRA-treated cells. Time-course experiments with 1 muM ATRA showed enhanced PAI-2 mRNA **expression** as early as 2 h, reaching a maximum at 4-6 h and then declining at 18-24 h, and a time-dependent increase in PAI-2 antigen in the cell culture medium. At variance with PAI-2, u-PA was notinfluenced by the drug. To establish whether ATRA-induced changes influenced the fibrinolytic process, we evaluated the effect of MNC stimulated with ATRA on u-PA-induced degradation of diluted plasma clots. ATRA-treated cells markedly inhibited clot lysis induced by low concentrations of u-PA. The effect was due to enhanced extracellular PAI-2 accumulation since it was observed with conditioned medium from ATRA-treated cells; it was abolished by the addition of neutralizing anti-PAI-2 antibodies and was negligible when single-chain t-PA was used instead of u-PA. Since monocyte/macrophage-mediated, plasminogen-dependent extracellular proteolysis has been proposed as an important mechanism of tissue damage in several inflammatory states, our findings might contribute to better explain the anti-inflammatory properties of **retinoids** .